Valve Resistance

Lincoln E. Ford, MD; Ted Feldman, MD; John D. Carroll, MD

We recently proposed that hemodynamic resistance is a useful index of valvular stenosis, and there have since been additional studies investigating this index. The proven usefulness of calculated valve area in describing stenosis raises the important question of why such a proposal was made. The question is all the more pertinent because resistance is calculated from the same data as area. This editorial is an attempt to answer the question directly. In our view, the benefits of this index derive as much from the new ideas that emerge from the concept of resistance as from its greater accuracy in describing stenosis and predicting outcome.

We offer four reasons, described separately below, for proposing that resistance be used, at least as an adjunctive index, in describing stenosis: (1) the theoretical basis for the area calculation is incorrect; (2) resistance appears to remain more constant than calculated area when flow varies; (3) further hemodynamic calculations follow naturally from resistance but not from calculated area; and (4) resistance suggests further useful studies of stenotic valves, whereas calculated area does not. The first two of these reasons suggest that resistance might be a more satisfactory or a more accurate index of stenosis, and a recent clinical study showed that resistance gives a better measure of the severity of stenosis when cardiac output is low. We regard these as the less important reasons for our proposal. We believe that, despite its limitations, calculated valve area is a useful clinical index and should be retained. To us, the more important reasons for our proposal are the last two: in essence, that the use of resistance suggests future directions for hemodynamic investigations. A major point to be made is that resistance can be used to predict the relation between pressure gradient and flow across the stenotic valve. Whether resistance remains constant or not, a knowledge of the relation between pressure and flow can be very helpful in understanding the hemodynamic effects of stenosis.

Theoretical Considerations

The theoretical model on which the Gorlin's4 based their calculations is the Torricelli principle of laminar fluid flow through a flat orifice, depicted in the Figure (panel A). In this model, valve area (A) is calculated by the equation

$$A = F/[k \cdot (\Delta P)^{1/2}]$$

where F is the mean systolic flow, \(\Delta P\) the mean systolic pressure difference across the valve, and k a constant. If calculated area is to remain constant at different flow rates, the pressure must be proportional to the square of flow.

Shortly before the Gorlin's published their seminal paper, two other pioneering laboratories in cardiac catheterization suggested that valvular resistance be used as an index of stenosis. Resistance is defined simply as mean pressure difference divided by mean flow, ie,

$$R = \Delta P/F$$

and is analogous to electrical resistance in Ohm's law (E=IR, R=E/I). If resistance is to remain constant with changes in flow, pressure must be proportional to the first power of flow. Thus, the major difference between the area and resistance indices is that the former implies that pressure is proportional to the square of flow and the latter implies that it is proportional to the first power of flow.

The Gorlin's and others referred to the resistance concept as the Poiseuille model, because in this model, pressure is also proportional to the first power of flow. As illustrated in panel B of the Figure, however, the Poiseuille model of laminar flow through a tube of uniform cross-sectional area is not appropriate to the stenotic valve, which is more accurately described as turbulent flow through a tapered tube (Figure, panel C).

Neither the Torricelli nor the Poiseuille model accurately reflects true valvular flow, primarily because flow downstream from the valve is turbulent and not laminar. A conceptual advantage of the Torricelli model is that it does not require estimation of valve length, as would the Poiseuille model, or any rigorous physical description of the valve. Possibly the lack of a need to measure valve length contributed to the ready acceptance of the Gorlin formula for calculating area. Whatever the reason, the relatively simple concept of planar valve area, described in dimensions of square centimeters, came into general usage, whereas the concept of resistance, with its arcane units of dyne/sec/cm^2, fell into obscurity. In our view, the consideration that the theoretical basis for calculating area is incorrect makes this a somewhat less desirable index. By contrast, the calculation of valve resistance requires no theoretical framework; it is simply a statement of how valve flow and pressure gradient are related.

Constancy of Resistance

Several recent studies have reevaluated the Gorlin formula and found that calculated areas increased as flow increased. These increases occurred because flow was more nearly proportional to the first power rather than the square root of the pressure gradient. The Gorlin's and others originally favored calculated valve area over the resistance index because it appeared to remain constant as flow rate varied. The more recent studies suggest that this reason is incorrect and that resistance appears to give a more constant description of the valve at different flow rates. In retrospect, it is possible that the pulmonary artery wedge pressures...
Further Calculations Using Resistance

Resistance is simply a statement of the relation between pressure gradient and flow across an obstruction, and as such, is a way of predicting one variable from the other. There is no a priori reason to believe that resistance will be independent of flow rate. Although it is convenient that resistance appears to be independent of flow, its predictive value would not be diminished if it were found to vary with flow under some circumstances, as long as the circumstances are known. When one of the two variables can be predicted from the other, it is possible to make a number of useful hemodynamic predictions.

A very simple and useful calculation that follows immediately from the predictive capability of resistance is the increase in pulmonary pressure that occurs in response to increased cardiac output in the presence of mitral stenosis. To the extent that pressure gradient varies directly with flow, the pressure across the valve will increase in direct proportion to flow. This consideration leads directly to an understanding of the debilitating effects of mitral stenosis. For example, when cardiac output doubles, a resting gradient of 15 mm Hg will double to 30 mm Hg, with a concomitant increase in the pulmonary capillary pressure.

Another simple and useful calculation is the workload imposed by a stenotic valve and the change in workload that occurs with a change in cardiac output. If the relation between resistance and flow is known, the alterations in pressure gradient and workload imposed by a stenotic valve at different flow rates can be calculated immediately. Furthermore, the workload imposed on the ventricle can be compared with the workload imposed by the periphery, and the fraction of the total ventricular work imposed by the valve can be measured. In our previous article, we made calculations of this sort to illustrate their usefulness in assessing the functional effects of stenosis. In another set of calculations, we showed how the pulsatile nature of a purely systolic load increases ventricular work in inverse proportion to the fraction of the cardiac cycle spent in

used by the Gorlins to estimate valve pressure gradients were damped or hybrid pulmonary artery pressures. These pressures can vary significantly with cardiac output in patients with mitral stenosis.

It would be very difficult to make any theoretical prediction of the pressure-flow relations of the diseased valve depicted in the Figure (panel C). The complexity of the physical conditions suggests that this relation is best determined empirically. Since the murmurs heard in valvular stenosis imply turbulence, and since turbulence increases resistance to flow, it might be expected that the pressure gradient across the valve would rise out of proportion to flow, as suggested by the Gorlins. The observation that it does not is surprising, and three possible explanations have been offered: (1) the valve dilates so that physical area is increased at high flows; (2) the blood viscosity decreases with turbulent flow; and (3) because of Venturi effects, the pressure immediately distal to the valve is lower than the pressure farther downstream, so that measurements made immediately distal to a valve may overestimate the gradient. The mechanisms invoked by the first two of these explanations are not mutually exclusive. In addition, diseased valves may differ substantially in their physical characteristics such that one mechanism may predominate in some valves and another mechanism may predominate in others. Whatever the mechanism, empirical observation indicates that resistance remains more constant than area as cardiac output varies and may thus be a more reliable indicator of hemodynamic impairment.

This greater reliability has been further shown by a recent study indicating that calculated area overestimates the severity of stenosis when cardiac output is low.

We should emphasize that our view of the importance of the concept of resistance does not depend on the resistance remaining constant. If resistance were found to vary with flow in some circumstances but not in others, it would be very helpful to know the factors that determine the dependence on flow. As explained below, the hemodynamic severity of stenosis may depend in very large part on the way resistance varies with changes in flow.
systole. These latter calculations further illustrate how arterial compliance reduces ventricular power requirements by spreading the work over the cardiac cycle. In a final set of calculations, we showed how the stenotic valve absorbs a greater fraction of the myocardial work at higher cardiac outputs, thereby limiting the cardiac reserve substantially.

The main purpose of all of these calculations was to show how resistance leads naturally to these hemodynamic calculations, whereas the area index does not. The static, anatomic index of valve area has virtually no predictive value in hemodynamic calculations.

Future Studies Using Resistance

Estimates of valve area from hemodynamic data lead naturally to correlations with other anatomic measurements made in other ways, such as valve imaging, but these measurements are difficult to correlate with functional impairments. By contrast, an exact knowledge of the relation between pressure and flow under different conditions could form the basis of many fruitful hemodynamic investigations. In addition to validating the types of calculations described above, variations of resistance under different conditions might lead to the recognition of previously unknown distinctions in the types of stenosis. As mentioned, the finding that valve resistance remains relatively constant as cardiac output increases was somewhat unexpected and has suggested to some authors that the physical valve area expands with an increased pressure gradient. To the extent that a valve dilates, it will impose less limitation on cardiac reserve than a noncompliant valve. By comparing the changes in resistance that result from varying flow rate with physical areas measured by some anatomic imaging, we might be able to distinguish variations in the severity of stenosis among valves having the same resistances and areas at rest. To do this, however, requires a method of measuring area that is independent of the hemodynamic measurements. By using hemodynamic measurements to calculate area, previous studies have obscured the possibility of disparities between anatomic and hemodynamic changes.

We have suggested that the increase in calculated area with increased cardiac output might result from a decreased blood viscosity at high flow rates and high turbulence. Such viscosity changes might result from changes in the aggregation of red blood cells as shear rates change. This suggestion leads to the question of the effects of hematocrit on resistance and on the possibility of both resistance and the variation of resistance with flow being altered by changes of hematocrit. Would it be possible, for example, to increase cardiac output by decreasing hematocrit? If this were the case, there might be an optimum hematocrit for oxygen delivery to the heart.

Another large area of investigation would be the way in which the ventricle responds to increased workload in the presence of stenosis. As we have described earlier, this will depend very much on whether ventricular performance is limited by the pressure the ventricle can generate or by the rate at which it can work. Again, a variety of factors, such as the adequacy of coronary reserve or the degree of hypertrophy, may cause different ventricles to respond differently.

The main point of this discussion is not to suggest which areas should be pursued, but simply to illustrate the wide range of studies that might be suggested by a hemodynamic index of stenosis.

Conclusions

The principal value of reporting stenosis in terms of hemodynamic resistance rather than anatomic area is that it is likely to focus attention on functional rather than anatomic consequences. Resistance has the additional benefits that it appears to change less with cardiac output than calculated area and that it is not based on any unwarranted physical assumptions.

References


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